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Research report

An investigation of facial emotion recognition impairments in alexithymia and its neural correlates

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HIGHLIGHTS

- We examine neural correlates of emotion recognition impairments in alexithymia.
- High degree of alexithymia is associated with impaired facial emotion recognition.
- High degree of alexithymia is associated with less activity in ACC and other regions.
- High alexithymia is associated with more activity in the superior parietal lobule.

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ABSTRACT

Alexithymia is a personality trait that involves difficulties identifying emotions and describing feelings. It is hypothesized that this includes facial emotion recognition but limited knowledge exists about possible neural correlates of this assumed deficit. We hence tested thirty-seven healthy subjects with either a relatively high or low degree of alexithymia (HDA versus LDA), who performed in a reliable and standardized test of facial emotion recognition (FEEL, Facially Expressed Emotion Labeling) in the functional MRI. LDA subjects had significantly better emotion recognition scores and showed relatively more activity in several brain areas associated with alexithymia and emotional awareness (anterior cingulate cortex), and the extended system of facial perception concerned with aspects of social communication and emotion (amygdala, insula, striatum). Additionally, LDA subjects had more activity in the visual area of social perception (posterior part of the superior temporal sulcus) and the inferior frontal cortex. HDA subjects, on the other hand, exhibited greater activity in the superior parietal lobule. With differences in behaviour and brain responses between two groups of otherwise healthy subjects, our results indirectly support recent conceptualizations and epidemiological data, that alexithymia is a dimensional personality trait apparent in clinically healthy subjects rather than a categorical diagnosis only applicable to clinical populations.

1. Introduction

Alexithymia describes difficulties to identify and describe feelings, a functional and externally oriented way of thinking and reduced emotional awareness in interpersonal interactions. Originally coined in the 1970s, the term alexithymia was introduced as a typical trait in psychosomatic patients, complaining about multiple somatic symptoms due to the lack of a symbolic language to explain their feelings [1]. In the original view, alexithymia represented a categorical diagnostic entity equalling a clinically relevant condition. On the other hand, recent conceptualizations and epidemiological data support the notion that alexithymia is a dimensional personality trait showing a normal distribution in the general population with significantly higher levels in male subjects [2]. Although alexithymic traits can be found in healthy subjects, high levels of alexithymia still represent an independent risk factor for different medical and psychiatric conditions and can best be viewed within the framework of dysfunctional emotion regulation

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and recognition [3,4]. Empirically though, the latter aspect – impaired recognition of others' emotions in alexithymia – is still discussed controversially. The major instrument to measure alexithymia is the Toronto Alexithymia Scale (TAS-20), which relies on self-rating of a subject's ability to identify and describe feelings [5]. It is not clear, to what extent this self-rating correlates with objectively measured capacities to actually recognize emotions in others [6] and empirical studies showed mixed results [7]. Since the recognition of emotions from *facial expressions* plays an important role in interpersonal communication and is well-studied on the behavioural and neuronal level, we will focus on this aspect. A recent review argues that alexithymia is actually linked with deficits to recognize facially expressed emotions in healthy as well as clinical groups [8]. It is important to notice that although there are studies showing impaired emotion recognition from faces in healthy subjects with alexithymic features [9–12], others have shown no such correlations [13–16]. Interestingly, the only study controlling for verbal abilities found no significant differences in facial emotion recognition between healthy subjects relatively high or low in alexithymia [17]. Even in research with various patient groups with clinically assigned alexithymic features the picture is heterogeneous [18–22].

Regarding this controversy, it is of interest if subjects with alexithymic traits show differential recruitment of brain areas associated with emotion processing when confronted with facially displayed emotions. The literature influencing our hypotheses can roughly be divided into three types of neuroimaging studies: emotion processing (across various tasks) in alexithymia, studies investigating the concept of emotional awareness and facial recognition in general. As for the neural correlates of emotion processing in alexithymia, various single studies and a recent meta-analysis will be reported. Presenting masked emotional faces to healthy subjects with varying degrees of alexithymia in the fMRI, the study of Reker et al. [23] showed activity in insula, superior temporal gyrus, middle occipital gyrus and parahippocampal gyrus to correlate with alexithymia. The study of Duan et al. [24] presented surprised faces subliminally and found activity in parahippocampal gyrus and fusiform gyrus. Finally, the studies of Eichmann et al. [25] and Kugel et al. [26] found a negative correlation between the degree of alexithymia and activity in the fusiform gyrus [25] and the right amygdala [26] when confronted with masked sad facial expressions. The recent meta-analysis by van der Velde et al. [27] examined 15 studies across various task types and valence of emotions and provides converging evidence of a relative hypofunction in alexithymia in amygdala, fusiform gyrus, premotor areas, dorsomedial prefrontal cortex (dMPFC), Insula and precuneus. An interesting case is the activity of the anterior cingulate cortex (ACC) in alexithymia. ACC hypofunction has been associated with alexithymia in many neuroimaging studies (e.g. [28–30]) and is also evident in reduced emotional awareness (see below). In contrast though, the meta-analysis by van der Velde et al. [27] reported a relative ACC *hyper*function in alexithymia. This discrepancy will be considered in the discussion. On the methodological side, two recent meta-analyses show that the majority of alexithymia neuroimaging studies do not explicitly assess the ability to actually recognize emotions [8,27] when using emotional faces as stimuli but rather present faces subliminally. Additionally, only a limited set of emotions was typically used in each study (mostly only up to three). Since we were interested in explicit recognition of a representative array of emotions (i.e. the six basic emotions), previous studies are difficult to compare. On the other hand, the review by van der Velde et al. reported activity independent of task type, which encourages us to derive hypotheses regarding brain areas with hypofunction in alexithymia from the above mentioned literature.

A second group of studies guiding our hypotheses is centred around the concept of emotional awareness, which is a type of cognitive processing of emotions undergoing five levels in rising order [32]. Deficits in emotional awareness are part of the broader concept of alexithymia but not identical to it. This theoretical distinction is supported by empirical findings showing limited correlations between the two concepts (e.g. [33,34]). Many neuroimaging studies of emotional awareness use measures of subjective attention to feelings by studying affective films and pictures. Within this experimental framework, recent neurobiological models posit a deficit of the anterior cingulate cortex (ACC) in the processing of emotions [35,36]. Accordingly, in the general population, the central role of the anterior cingulate and medial prefrontal cortices in emotion processing has been well established [37,38]. Therefore, despite the reported ACC hyperfunction in alexithymia in the meta-analysis by van der Velde et al. [27], we still hypothesize less ACC activation in alexithymia derived from its generally important role in emotion processing [38], the concept of its functional deficits in reduced emotional awareness [39], and neuroimaging studies of alexithymia showing ACC hypofunction (e.g. [28–30]).

The last body of literature concerns the neural correlates of facial recognition in general. The most influential model by Haxby et al. proposes both, a core and an extended system [40,41]. The core system (occipital face area, fusiform face area and posterior superior temporal sulcus) is involved in basic face processing (independently of emotional content) and hence not probable to show strong abnormalities in alexithymia. Of more interest for our study is the extended system which is primarily concerned with extracting meaning from faces, i.e. all the aspects of social communication and emotion. Areas of the extended system processing facial emotions include amygdala, insula and striatum [40,41]. Additionally, the inferior frontal gyrus (IFG) [42] and thalamus as a "sensory gateway" [43] have been implied in emotion recognition from faces. Finally, in an extensive meta-analysis of over 100 studies comparing processing of emotional versus neutral faces, Sabatinelli et al. [44] showed emotion-specific activity in the amygdala, fusiform gyrus, medial prefrontal cortex (mPFC), inferior, superior and middle frontal gyrus, parahippocampal gyrus and middle temporal gyrus.

From this background, we searched for neural correlates of hypothesized deficits in facial emotion recognition in alexithymia. To this end, we assessed the ability to recognize facially expressed emotions with a standardized and reliable test system using functional magnetic resonance imaging (fMRI) in two groups: healthy subjects with relatively high (HDA) or low degree of alexithymia (LDA). The discrepancy between the modern concept of alexithymia as a dimensional trait and our methodological approach categorizing subjects into HDA and LDA merits some explanation: On the one hand, we do believe that alexithymia is a dimensional trait that is present in the general population. In order to avoid confounding factors such as psychopathological symptoms (e.g. anhedonia in depression), we deliberately chose to only recruit healthy participants. On the other hand, we opted for a between-group design comparing two relative extremes within the healthy subjects to improve testing of differences in brain activity. Although this categorizes alexithymia again, it is done within a non-clinical group and for the sake of hypothesis testing. Our methodological decision is backed by the approaches apparent in previous research: Six out of 15 studies of emotion processing in alexithymia mentioned in the meta-analysis by van der Velde et al. [27] use this approach contrasting high versus low alexithymia subjects, and in all of those studies "healthy" participants without any clinically relevant psychopathological symptoms were investigated.

We hypothesized that HDA subjects would perform worse in the facial emotion recognition task compared to the LDA group and that HDA subjects would show differential neuronal activity in key brain